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Editorial

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Why are we not working to prevent OSA?

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As healthcare professionals and health scientists, our responsibility extends beyond diagnosis and treatment to efforts to prevent disease where possible. In this issue of *Sleep*, Correa et al [1] make a "call to action" on the prevention of a very common sleep disorder, obstructive sleep apnea (OSA). The case for prevention, as outlined by the authors, is indeed compelling. OSA is highly prevalent globally and has been consistently linked to wide-ranging adverse health outcomes, including impaired growth and development in early ages of life [2], motor vehicle and work-related accidents, impaired cognitive function and quality of life, and increased rates of cardiovascular disease and overall mortality [3, 4]. The related estimated economic costs are large and the currently available resources needed to diagnose and treat OSA are inadequate. OSA is greatly underdiagnosed but even when diagnosed and treated adherence to treatment is often suboptimal [5]. Overall, the potential benefit from even small successes in preventing OSA would appear to be substantial.

To better understand the state of knowledge on the prevention of OSA, the authors undertook a systematic review to identify relevant articles in the literature. Their search criteria resulted in 720 potential articles on prevention. By contrast, there were many more articles found, using comparable search terms, focused on diagnosis (17 382) and therapeutics for OSA (23 647). After further screening and review, there were only 4 relevant articles identified. Some of the review's inclusion/exclusion criteria [1], appear to be quite restrictive, partly accounting for the very low number of four papers identified at the end of the process. For example, articles were included if they were considered to be "Preventive OSA medicine" and excluded if there was "No reference to proposed action." Consequently, many articles reporting OSA risk factors were excluded as they did not meet the above criteria, yet may contain useful information relevant to the prevention of OSA. The authors acknowledge there is a well-established literature on risk factors but conclude that their findings highlight the relative dearth of literature specifically focused on preventative strategies. In view of the limited research on OSA prevention, it is not surprising that little attention is paid to its prevention in any of the major scientific sleep societies' guidelines. Nor is there widespread public health messaging about measures to reduce the risk of developing OSA.

It is indeed notable that no interventional studies for the prevention of OSA were identified in the systematic review. The authors cite a paper by Liu et al [6] on risks for incident OSA, which found self-report sedentary behavior and lower levels of physical activity were associated with a higher incidence of OSA during 2 004 663 person-years of follow-up, but note limited other data on the incident disease. In the absence of adequate data on preventative approaches, the authors discuss a number of anticipated modifiable OSA risk factors (obesity, inactivity, alcohol consumption, and poor diet) that warrant targeting given their established associations with the presence and severity of OSA. Obesity is the strongest of these, with a 2018 American Thoracic Society guideline concluding that randomized trials of weight loss interventions, such as low-calorie diet, exercise, behavioral interventions, and combinations of these, lead to improvements in OSA severity, albeit with generally low certainty in their estimated effects [7]. Unfortunately, public health campaigns targeting weight loss have generally had low success and, hence, the authors suggest a need for collaborative public health campaigns with the inclusion of measures of OSA emergence to assess the effect of such campaigns on incident OSA. There is good evidence suggesting that exercise reduces the severity of OSA independent of its effects on weight, perhaps due to lowering inflammation and ameliorating body fluid shifts [8–10]. The few data on diet quality and OSA are largely cross-sectional and show associations between low diet quality and the presence or severity of OSA [8]. Unfortunately, while the benefits of a healthy lifestyle on a range of chronic illnesses, such as cardiovascular and metabolic diseases, are well known, the relevance in terms of prevention of OSA appears to be rarely, if ever, communicated in public health campaigns.

We concur with the authors that early childhood identification of maxillofacial risk factors, such as prematurity, retrognathia, high arched palate, and midface hypoplasia, is important as structural and functional orofacial development plays a key role in prevention of OSA. More research is needed to validate

simple tools that can assist in the identification of the presence and severity of these orofacial features, including 3D facial photography, intra-oral scanning, and prenatal facial scanning [11–13]. Education of primary care physicians and dentists to identify these risk factors [14] will assist in the development and implementation of prevention strategies.

A developmental origin of health and disease framework [15] advocates for the prevention of disease to start from birth or even before, in the preconception period. Although the evidence is limited, some early life risk factors affecting orofacial development could be modifiable. Examples include preventing premature birth, no alcohol use in pregnancy, breastfeeding, and selective treatment of short frenulum, which encourage orofacial development [16–18] by various physiological mechanisms [19]. However, some risk factors for orofacial development are largely nonmodifiable since they are related to genetic and familial predisposition [19, 20], such as craniofacial dysmorphology, retrognathic mandible, floppy upper airway, or other associated syndromes [20]. In these situations, prevention means preventing neurological, cardiovascular, or metabolic comorbidities by early recognition and treatment of OSA. Future research to explore modifiable prenatal and postnatal risk factors causing upper airway dysfunction in the fetal or neonatal period to prevent OSA is needed.

There are many examples in the literature of proposed preventative health measures, which showed great promise according to observational data, yet their effectiveness was not borne out in subsequent randomized trials. A large part of the explanation for such an apparent discrepancy appears to be a failure to account for healthy-user bias in observational studies such that the effect of the risk factor of interest is overestimated [21]. Hence, there is clearly a need for interventional trials of preventative approaches targeting well-established risk factors for OSA. There may be considerable challenges in designing interventional trials and the authors do not provide guidance in this regard. However, encouragement for the potential value of such studies can be taken from the cardiovascular field, where measures to mitigate modifiable risk factors have been shown to reduce the development of major adverse cardiovascular events. For example, strong evidence from numerous randomized trials shows that reduction of blood pressure to target levels in various populations reduces the risk for CVD events and all-cause mortality by 20%–40% [22, 23].

There are some signs of a shift occurring toward a more preventative approach to sleep disorders, including OSA. In Australia, spurred by advocacy from the Australasian Sleep Association, the Sleep Health Foundation, and others, the Australian government has invested in policy reviews resulting in a range of preventative sleep health strategies focused on the primary health care setting [24] and have embedded sleep health in its National Preventative Health Strategy 2021–2030. We hope that the article by Correa et al [1] stimulates further discussion which leads to action on the neglected topic of preventing this common and impactful sleep disorder.

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